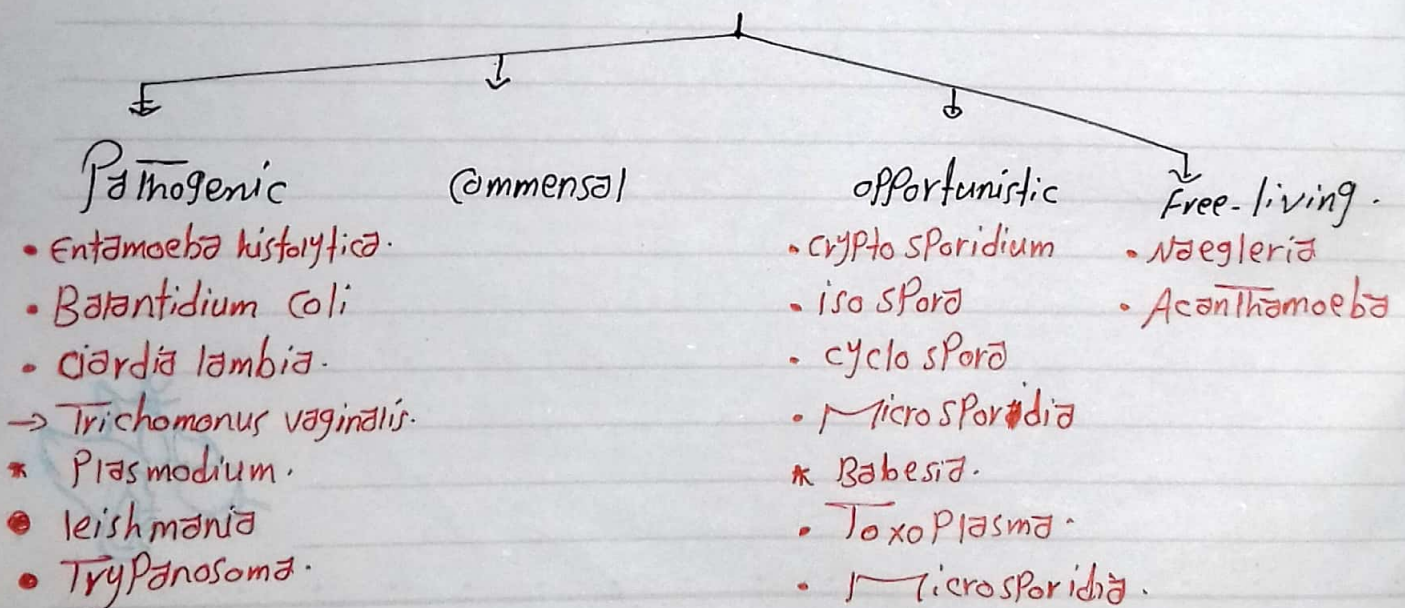


بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

Protozoa

Classification of Protozoa according to Their effect on body of The Patient :-

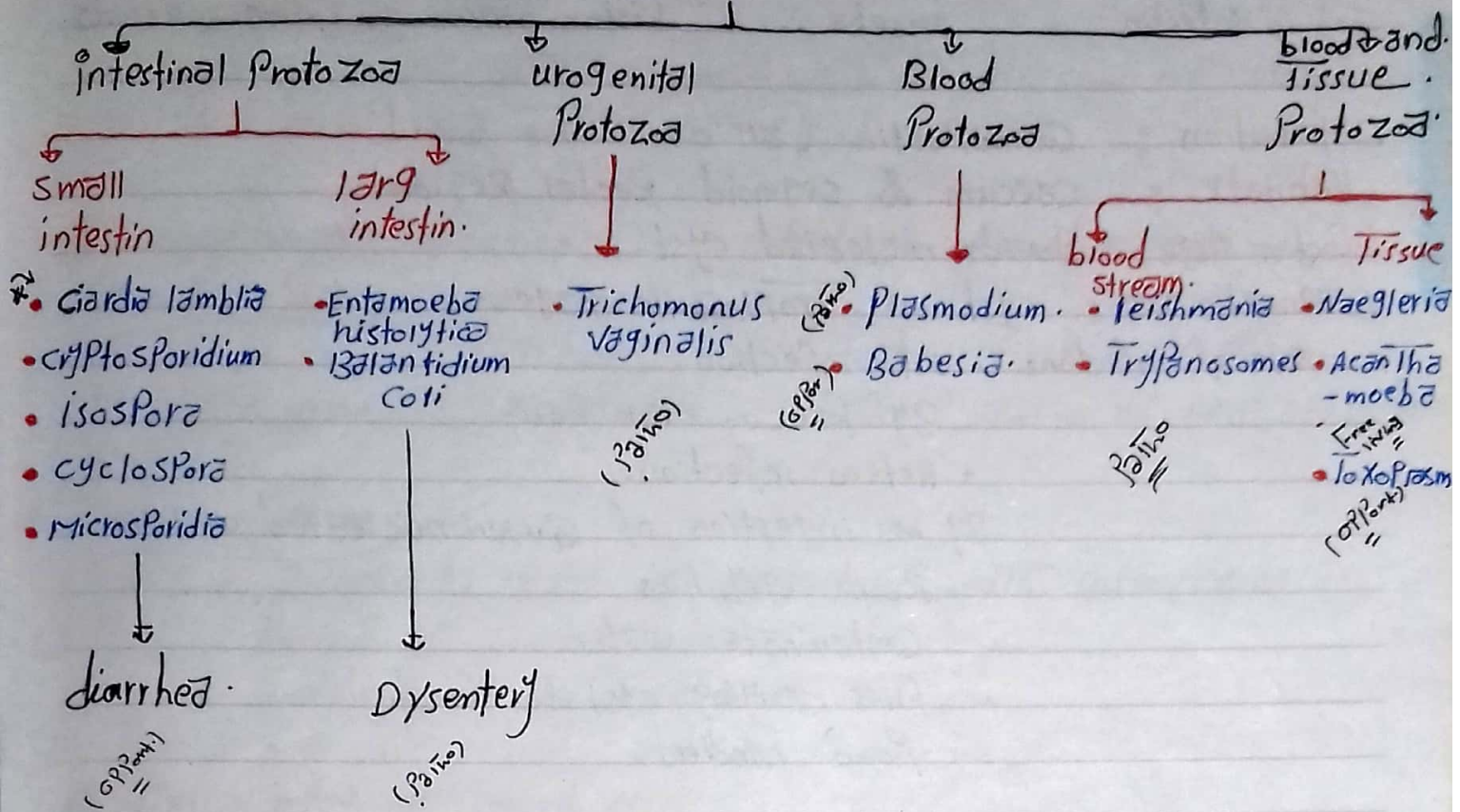
- **Pathogenic Protozoa** → Protozoa That exist in human body cause harm to infected human.
- **Commensal Protozoa** :- Protozoa That exist in human body but doesn't cause harm to infected human.
- **Opportunistic Protozoa** → weak Protozoa That causes minimal effect to infected healthy human. but several effect. To infected immuno compromised human.
- **Potentially Pathogenic free-living Protozoa** → Free living in nature away from man but some of them may cause disease if they enter human body by certain Route.



VISIT...

LANZAROTE
Caliente.COM

Classification of Protozoa according to Habitate.



U-13 blood & tissue. Protozoa — Protozoa that swim in. Patients blood stream then attack his tissues.

⇒ Protozoa affecting different system — Microsporidia.

Trophozoite stage
cyst ~

Sporozoite stage
oo cyst stage.

Entamoeba histolytica.

Ent → intestin amoeba histo → tissue lytica → dissolve

Distribution :- Cosmopolitan (all over the world).

Habitat :- caecum & sigmoid - Rectal Region.

Infective stage :- Quadri-nucleated cyst.

Diagnostic stage :- cyst or Trophozoite stage.

Mode of infection :-

- auto infection.

By faeco-oral Route.

- Hetero infection.

By ingestion of Quadri-nucleated cyst in

- ✓ Raw vegetables

- ✓ Contaminated water

- ✓ Flies carrying cyst to food.

- ✓ Food handlers.

Pathogenesis:-

Pathogenic activity depend on.

- 1- Parasite virulence

- 2- Host Resistance.

- 3- Condition of intestinal Tract.

→ Parasite may remain in intestinal lumen. so called.
Non Pathogenic.

→ may secrete histolytic enzyme. Produce necrosis in intestinal mucosa leading to formation of flask-shape ulcer. This followed by.

- ①- Proliferation of C.T around ulcer → Thickening of wall
- ②- intensive ulceration may be accompany by 2nd infection
- ③- Extra intestinal invasion to brain, liver, lung, skin.

Clinical Picture :-

① Asymptomatic infection.

Parasite found in the lumen. without invasion of mucosa and cyst Pass in Stool (cyst Passer)

② symptomatic infection: "intestinal Amoebiasis."

Acute stage;

- * fever, diarrhoea, dysentery, abdominal Pain.
- Tenderness, Tenesmus, Painful, spasm of anal sphincter.

chronic stage;

- * Recurrent attack of dysentery. with intervening period of constipation.
- * weight loss and cachexia

③ Extra intestinal amoebiasis.

Hepatic Amoebiasis

- Enlarged Tender liver with Pain in Rt. hypochondrium.
- Amoebic abscess.

Pulmonary Amoebiasis.

- Consolidation.
- Trophozoites may appear in sputum.
- lung Abscess.

~~Complication~~ :-

- 1- Amoebic hepatitis → Pain in Rt. hypochondrium and Enlarged Tender liver.
2. Amoebic Abscess → fever, leucocytosis, Enlarged tender liver
- 3- Pulmonary Amoebiasis → fever, leucocytosis, Evidence of Consolidation, lung Abscess., may erode bronchus and Trophozoite appear in sputum.

sheet **Complication:-**

1- Amoeboma → granuloma around the ulcer
Tumor like mass.

2- Perforation of ulcer → Peritoneal infection.

3- Haemorrhage due to erosion of blood vessels.
in intestinal wall.

4- Stricture due to healing by fibrosis.

5- Appendicitis.

Diagnosis:- (intestinal Amoebiasis).

① **Clinically**: Painful frequent evacuation of small content
of stool containing mucus tinged with blood

② **Laboratory**.

* Direct Stool examination:-

sheet

Typical amoebic dysentery.

- * Stool bulky.
- * Acidic
- * scanty exudate.
- * Small amount of pus
- * blood.
- * Charcot-Leyden crystals.
are present
(degenerated eosinophils).

Typical Bacillary dysentery.

- * stool scanty.
- * Alkaline.
- * Massive exudate.
- * large amount of pus.
- * blood.
- * Charcot-Leyden crystals
are present

① examination of Trophozoites:-

- wet Preparation → highly refractile shining bodies with progressive directional crawl and ingested Red blood cell.
- Iodine stained. will reveal the morphology.
- Permanent stain Preparation by Trichrome stain or hematoxylin

② Examination of cyst Stained by iodine or Trichrome.

* Indirect Method:-

- serological Tests:- by ELISA
- Molecular Techniques
- Detection of Copro - Antigen in stool

* Radiological Examination using barium enema.

* Sigmoidoscopy. → see ulcer, Take biopsy.

Diagnosis Extra intestinal Amoebiasis.

Clinical :- according to organ affected

Laboratory:-

- Aspirate examination. from liver or lung abscess for Entamoeba Trophozoites.

- Serological test
- Radiology (elevated diaphragm.)
- leukocytosis due to 2nd infection.

Treatment :-

intestinal Amoebiasis :-

1. Metronidazole (Flagyl), Tinidazole (Fasigyn)
(very effective in killing of amoeba in wall of intestine, in blood, in liver abscess.) "Tissue amoebicide"

Diloxanide furoate.

(Kill Trophozoites and cysts in the lumen of intestine.
"luminal amoebicide".

Asymptomatic.



are given luminal amoebicide.
as Diloxanide furoate.

Symptomatic.



are given ^{Tissue} amoebicidal as
Metronidazole followed
by luminal amoebicide as
Diloxanide furoate.

Extra intestinal Treatment :-

- * Aspiration of Abscess.
- * Surgical drainage of Abscess.

Control

- * Treatment Patient.
- * Examination and treatment food handlers.
- * Environmental Sanitation.
- * Personal Prophylaxis.
- * Human faeces should not be used as fertilizer.

Balantidium coli

Distribution :- in Pige Raising area. "Commensal".

Reservoir host :- Bigs.

"in. intestine of big."

Infective stage :- cyst stage. ~ Balantidium coli cyst "

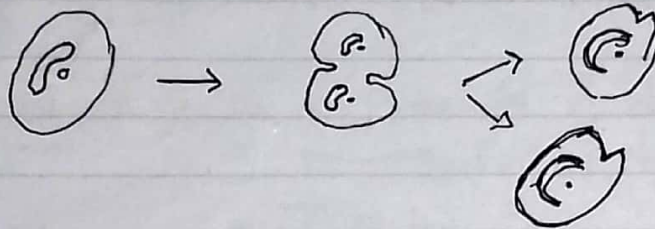
defentive host :- large intestine especially caecum.

Mode :- as Entamoeba.

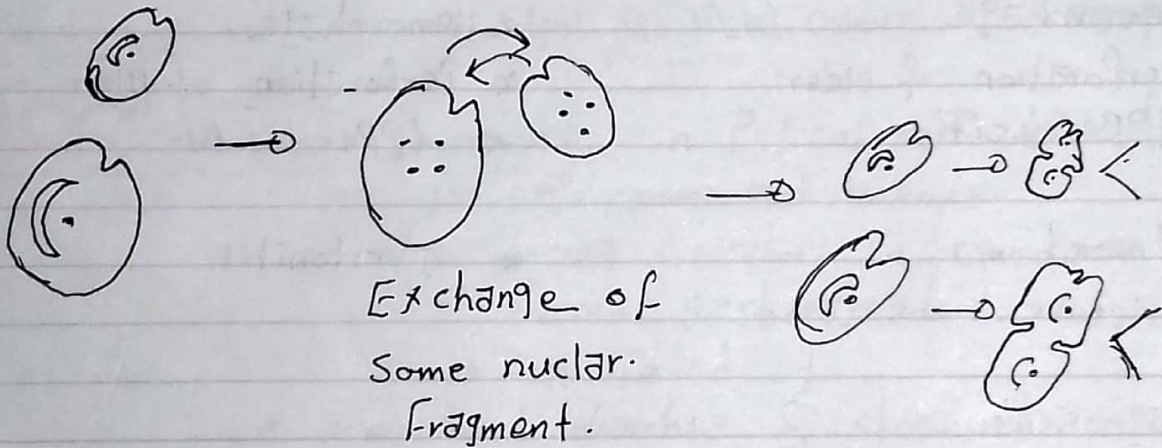
Diagnostic stage :- Trophozoite or cyst.

Multiplication of Balantidium coli

[1] Trophozoites multi by. Transverse binary fission.



[2] Conjugation between large and small Trophozoites.



dysentery :- Painful frequent evacuation of small quantities of stool. Containing mucous tinged with blood and tenesmus.

strain during defecation stool soft and watery.

Pathogenesis:

- 1- invasion of mucosa is affected by
 - Cytolytic Enzyme (hyaluronidase Enzyme)
 - Boring Action of cilia.
- 2- Secondary bacterial infection may follow the invasion. This leads to formation of flask shape ulcer.

*** 3- No Extra intestinal spread.

Clinical Picture:

sign and symptom of dysentery.

Complication:

Amoebiasis.

- * Hemorrhage.
- * Perforation of ulcer
- * Appendicitis
- * Amoeboma
- * Stricture of colon.

Balantidiasis.

- * Hemorrhage.
- * Perforation of ulcer.
- * Appendicitis
- * Peritonitis.

Diagnosis:

- * Clinically → Clinical manifestation of dysentery.
- * Laboratory →
 - * finding of Trophozoite or cyst in stool.
 - * Stool should be examined several times.
 - * Discharge of parasite is intermittent.

Treatment → Metronidazole.

← More common in children.

" Giardia lamblia "

Disease :- Giardiasis.

Distribution :- Cosmopolitan.

Habitat :- duodenum, upper part of small intestine.
bile duct, gall bladder.

Diagnostic stage :- cyst or Trophozoite stage.

Infective stage :- cyst stage.

Mode of infection :- As Entamoeba.

Pathogenesis and clinical picture :-

(1) **Asymptomatic** :- If parasite just feed on mucosa.

(2) **Symptomatic cases** :- * Hypaemia & duodenitis.
manifested by.

- Epigastric pain.
- disturbance in digestion.
- diarrhea & flatulence.
- Stool is light colour & greasy

(3) **Sever Symptom** :- occur in patient with impaired immunity
as 1- Hypo gamma globulinemia.
2- decreases secretory IgA in small intestine.
3- decreases gastric acidity.

manifested by.

- ✓ Persistent diarrhea & steatorrhea.
- ✓ Malabsorption.
- ✓ Hypo Proteinemia.
- ✓ decreases fat soluble vitamin.
- ✓ cholangitis & cholecystitis which may lead to jaundice & biliary colic.
- ✓ ~~by~~

Diagnosis :-

Clinically :- Clinical manifestation.

Laboratory :-

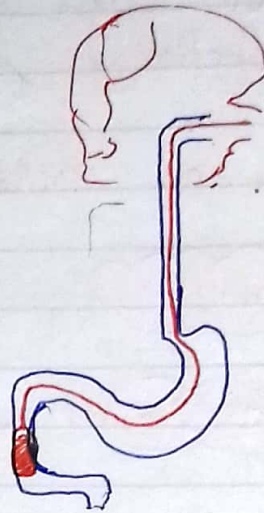
Direct examinations :-

- Trophozoite are detected in diarrhoeic stool
- cyst or trophozoite are detected in formed stool + chronic.
- Copro Antigen detection.

Indirect Through :-

- Serological Test. (little value).
- Detection of Copro Antigen.

String test :-



Treat with

Metronidazole.

leishmania and leishmaniasis.

Cutaneous

old world

- leishmania tropica.
- " major
- " aethiopica.

visceral.

old world

- leishmania donovani
- " infantum.

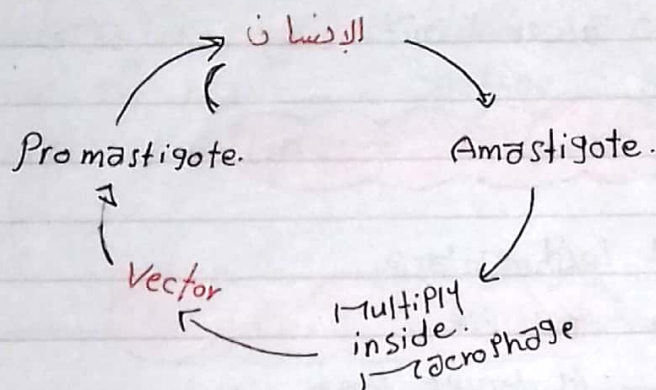
new world

- leishmania peruviana
- " braziliensis
- " mexicana.
- " pifanoi

new world.

- leishmania chagasi
- " amazona.

life cycle :-



vector :- sand fly.

old world cutaneous leishmaniasis.

- (1) leishmania tropica.
- (2) ~ major
- (3) ~ ethiopia.

R.H dog

leishmania tropica

sheet

"orient sore"

Pathogenesis:

- characterized by. Production of ~~sore~~ nodule. due to multiplication of organism in the skin macrophage.
- usually single.
- occurs primary in face.
- nodule is ulcerated.

sharp edge.
raised margin.
scanty exudate.

(it is called orient sore)

- Spontaneous healing without treatment leaving scar.
- 2nd bacterial infection is common. so parasite not seen on base of ulcer but only on edge.

R.H Rodents

leishmania major

(Rural, acute, moist lesion)

- it causes wet leishmaniasis.
- lesions are multiple.
- lesions occur in lower limb.
- ulceration occurs early.
- lesions moist with large exudate.
- Healing without treatment with scar formation.

- Face.
- single nodule.
- ulceration
- small exudate.
- dry ulcer.

- limbs.
- multiple lesion.
- ulceration.
- large exudate.
- wet ulcer.

leishmania aethiopica

- Produce diffuse cutaneous leishmaniasis. (without ulceration.)
- appear on face and exposed parts of limbs.
- like lepromatous leprosy.
- it Result from ① deficient cell-mediated immunity
② High virulence of parasite.

new world cutaneous leishmaniasis

- 1- leishmania Peruviana ~~~~~ like leishmania tropica.
- 2- " braziliensis.
- 3- " mexicana
- 4- " Pifanoi ~~~~~ like leishmania aethiopica.

R.H. Rodent
dog.

leishmania braziliensis

- ulcer Production in mucocutaneous area.
- ~~at~~ destruction of tissue, cartilage, bone.
- 2nd bacterial infection.

leishmania mexicana

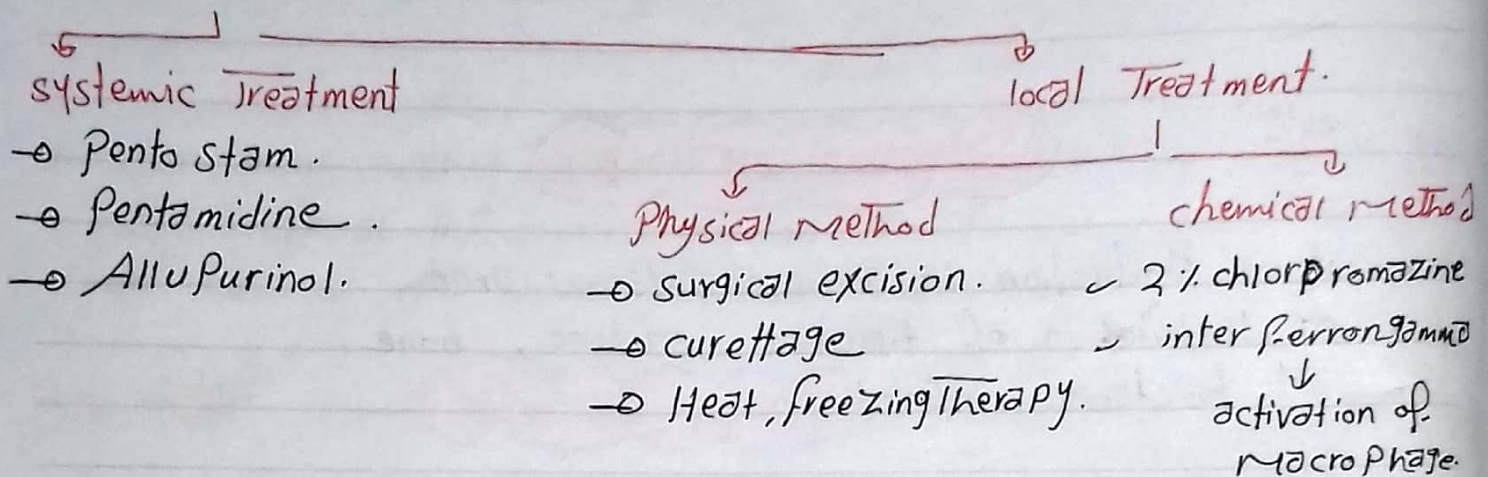
(chiclero ulcer)

- disease found on the area of chiclero plantation worker.
- it causes chiclero ulcer.
- it affect ear cartilage. causing its destruction.
- lesion single, painless and Heal spontaneously within 6 months.

Diagnosis of cutaneous leishmaniasis.

- (1) Clinical diagnosis.
- (2) Detection of Parasite.
 - * Sample is taken from edge of ulcer.
 - * Microscopic exam $\rightarrow$ amastigote.
 - * culture $\rightarrow$ Promastigote.
- (3) indirect diagnosis:-
 - * serological test or skin test (Montenegro test)
 - using antigen from cultured Promastigote.
 - Positive in 95% and remain positive for life.

Treatment:-



sheet

Visceral leishmaniasis. (Kala azar)

same

old world

1. leishmania donovani (R.E.S).
2. ~ infantum.

Pathogenesis :-

- sand fly bites man and inject promastigote, which are engulfed by ^{skin} macrophage and change into amastigote. That multiply and increase in number producing papule at site of bite (leishmanium).
- Parasite multiply in R.E.S of spleen, liver, bone marrow, intestine, L.N leading to bursting of these cells and liberate amastigotes invade other cell.

Clinical Picture.

- (1) leishmanium
- (2) Intermittent fever (double daily rise).
- (3) Hepato splenomegaly.
- (4) Pancytopenia.
- (5) Enlarged L.N
- (6) diarrhoea & dysentery.

sheet (7) skin change * During disease.

hyper pigmentation Dark pigmented area (black fever).

hypo pigmentation depigmented macule.

→ Butterfly distribution over nose.

Post kala azar dermal leishmaniasis.

→ Depigmented skin nodule ??

Due to

- affection of macrophage cell of skin.
- incomplet treatment

↓ Diagnosis :-

1- Clinical Picture in endemic area.

2- Direct diagnosis :-

Ⓐ blood film stained with leishman stain.

Ⓑ biopsy from spleen, liver, bone marrow.

Ⓒ Culture → promastigote.

Ⓓ Animal inoculation.

3- Serological tests.

Ⓐ Montenegro test.

* -ve during active kala-azar.
* +ve. after 2 months of
Successful Treatment.



Complication:- (causes of death). ?

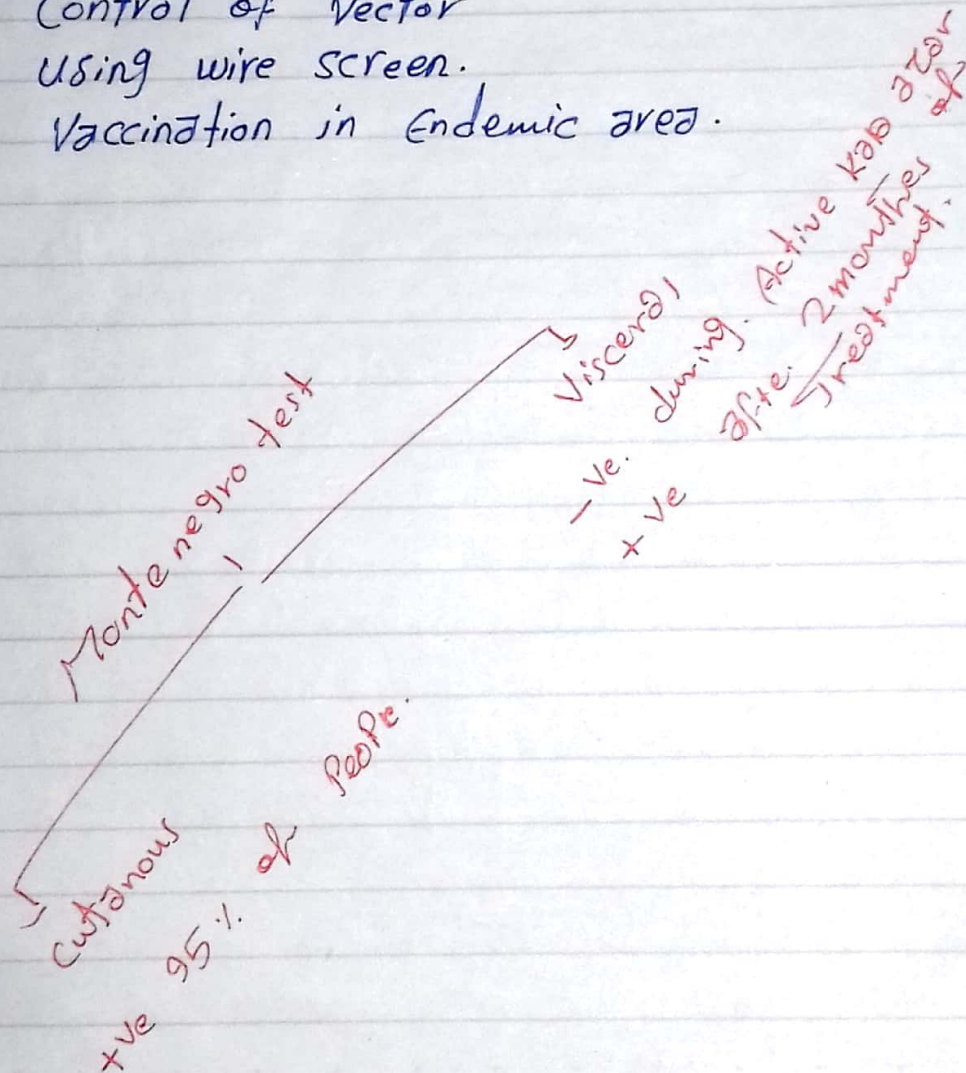
- 1- cell mediated immunity depressed. (suppression of cell mediated immune Response.)
- 2- 2nd infection.

Treatment:-

- ✓ 1- Antimony sodium gluconate (Pentostam).
- ✓ 2- Pentamidine
- ✓ 3- Allo Purinol.

Prevention & control.

- 1- Treatment of Patient.
- 2- Control of vector
- 3- Using wire screen.
- 4- Vaccination in endemic area.



Trichomonas vaginalis

disease :- Trichomoniasis.

Distribution :- Cosmopolitan.

Habitat :- Trichomonas vaginalis Trophozoite live in :-

→ Vagina and urethra of infected female.

→ urethra and Prostate of infected male.

infective stage :- Trophozoite stage.

diagnostic stages :- Trophozoite stage

Never become cyst

Mode of infection :- Transmitted directly during sexual intercourse from infected man to other.

Pathogenesis and clinical picture :-

(A) in women — **

Trophozoite found in vagina & urethra → feed on mucosal surface. → sloughing of ~~para~~ sq. Epith. cell.

● 50 % are asymptomatic.

● 50 % are symptomatic :-

✓ کثیر کی Profus odorous discharge.

✓ burning & itching.

✓ dyspareunia

✓ frequency of urination.

✓ dysuria.

کثیر کی

● on examination.

✓ vaginal wall inflammation.

✓ vulva inflamed.

✓ Excessive discharge.

⑬ In men *

Infection in male is commonly Asymptomatic. Symptoms appear when infection involve Prostate or higher parts of urogenital tract.

● Symptom include :

- ✓ Thin discharge
- ✓ dysuria (difficulty in urination).
- ✓ Nocturia
- ✓ epididymitis.

● On examination:-

Enlarged Tender Prostat. and Epididymitis.

Diagnosis :-

- ✓ (1) Microscopic examination of discharge.
 - ✓ vaginal
 - ✓ urethral
 - ✓ Prostatic.
 - ✓ urine sediment.
- ✓ (2) culture of discharge.
- ✓ (3) detection of Trichomonas vaginalis Antigen. by.
 - ✓ ELISA
 - ✓ Direct fluorescent Antibody test.
- ✓ (4) detection of DNA of Parasite. by P.C.R.

Treatment:- Metronidazole

- Treatment of sexual partner at same time.

~ Free living Amoeba "

They are amoeba that inhabit

- water (fresh, brackish and salty).
- Moist soil.
- Decaying vegetations . النباتات المألوفة

Potentially pathogenic free living Amoeba.

Naegleria fowleri
in water & air

Flagellated Form. ↔ Amoeboid Form (I.S.) ↔ cyst Form.

Acanthamoeba species.
in water & air

Trophozoite Form (I.S.) ↔ cyst form.

Naegleria fowleri

Flagellated
part
life cycle

Habitate :-
- water
- moist soil.
- ~~by~~ decaying vegetation.

Infective stage:- Amoeboid form.

Mode of infection:- Through nasal route during.

- 1- swimming in contaminated water
- 2- inhalation contaminated water.
- 3- inhalation contaminated air.

Diagnostic stage:- Amoeboid Trophozoite form.

Pathogenesis and Clinical Picture.

Pathogenesis :-

- Amoeboid Trophozoite invade nasal mucosa and cribriform plate and reach to brain along olfactory nerve.
- Naegleria Produce diffuse meningio Encephalitis with hemorrhagic inflammation and necrosis of brain tissue.

Clinical Picture :-

- 1- Fever & Headch & nausea & Vomting
Stiffness of neck & Convulsion.
2. disturbance in sense of smell or taste. occure.
- 3- coma and death occure within 3-6 days from infection.

Diagnosis.

as Granulomatous Acanthamoeba.

Treatment

- * There is no Compleet Treatmen.
- * Amphotercin B can be givein. I.v or intra Thecal

" Acanthamoeba species "

- Habitats:-
- water.
 - Moist Soil
 - decaying vegetation.

Infective stages- Acanthamoeba trophozoite.

Mode of infection.

Granulomatous

Amoebic Encephalitis.

- Trophozoite enter through nasal route → lower respiratory tract → blood → brain.
- Trophozoite enter through ulcerated skin or mucous → blood → brain.

Acanthamoeba

Keratitis.

- Trophozoite enter through.
 - ✓ Corneal trauma
 - ✓ exposure to contaminated water.
 - ✓ wearing contaminated contact lens.

Pathogenesis

Acanthamoeba causes single or multiple focal granulomatous lesions in brain.

Pathogenesis

Acanthamoeba causes.

- chronic progressive ulcerative keratitis
- corneal ulceration may progress to perforation.

Clinical Picture

- Headach, nausea, vomiting.
- ① - stiffness of neck and convulsion.
- ② Subacute or chronic course lasting to weeks or months or years.
- ③ in AIDS patient the disease may be fulminating.

Clinical Picture

- severe ocular pain.
- ① - affection of vision.
- in AIDS patient the infection may cause Endophthalmitis.

Diagnosis

1] History of Swimming.

2] C.S.F examination.

- Microscopic examination.
Reveals ^{Trophozoite} ~~amoeba~~ forms.
and cyst form

- suspension in fresh water
→ Transformation into
flagellate form.

- culture on suitable medium.

Diagnosis

Identification of trophozoite and
cyst in corneal scraping.
Then.

Examined directly under
microscope.
or

After culture.

Treatment

- * Excision of focal lesion.
+ Treatment with ketoconazole.

- * Penicillin & chloramphenicol.

Treatment

- * oral itraconazole.

+

Topical Miconazole.

- * Corneal Transplantation.

Naegleria meningio Encephalitis.

✓ Diffuse Meningio Encephalitis

✓ Rapidly fatal

✓ Nose → crural form

✓ children & young adult

Acanthamoeba Encephalitis.

focal granulomatous lesion in brain

Sub acute or chronic course.

lower Respiratory tract

old age, low immunity.

Plasmodium (Malaria)

	<i>P. vivax</i>	<i>P. oval.</i>	<i>P. Malariae</i>	<i>P. falciparum</i>
<u>Disease</u>	(vivax or benign Tertian malaria)	(ovartertian malaria)	(Quartan malaria)	(subtertian or malignant malaria)

Distrib. world wide. Tropical area Subtropical area Tropical area.
 (Presence of malaria = Presence of Anopheline Mosquito).

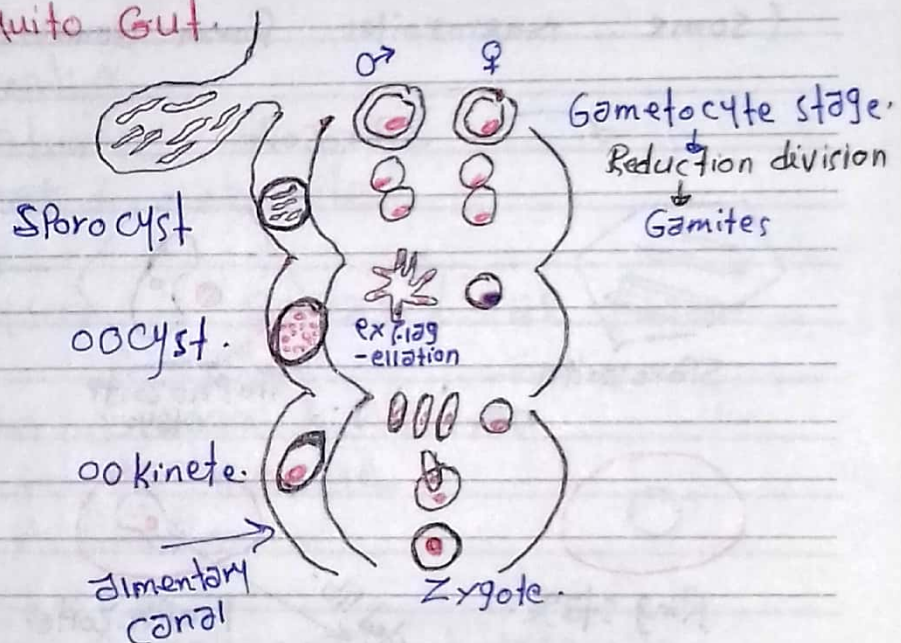
Infective stage Sporozoite stage.

Mode :
 • Through bite of female anophelous mosquito and inoculation of Sporozoite.
 • Sporozoite stage Through blood transfusion.

Diagnostic stage :-

Life cycle : Development of Plasmodium in Anopheles Mosquito Gut.

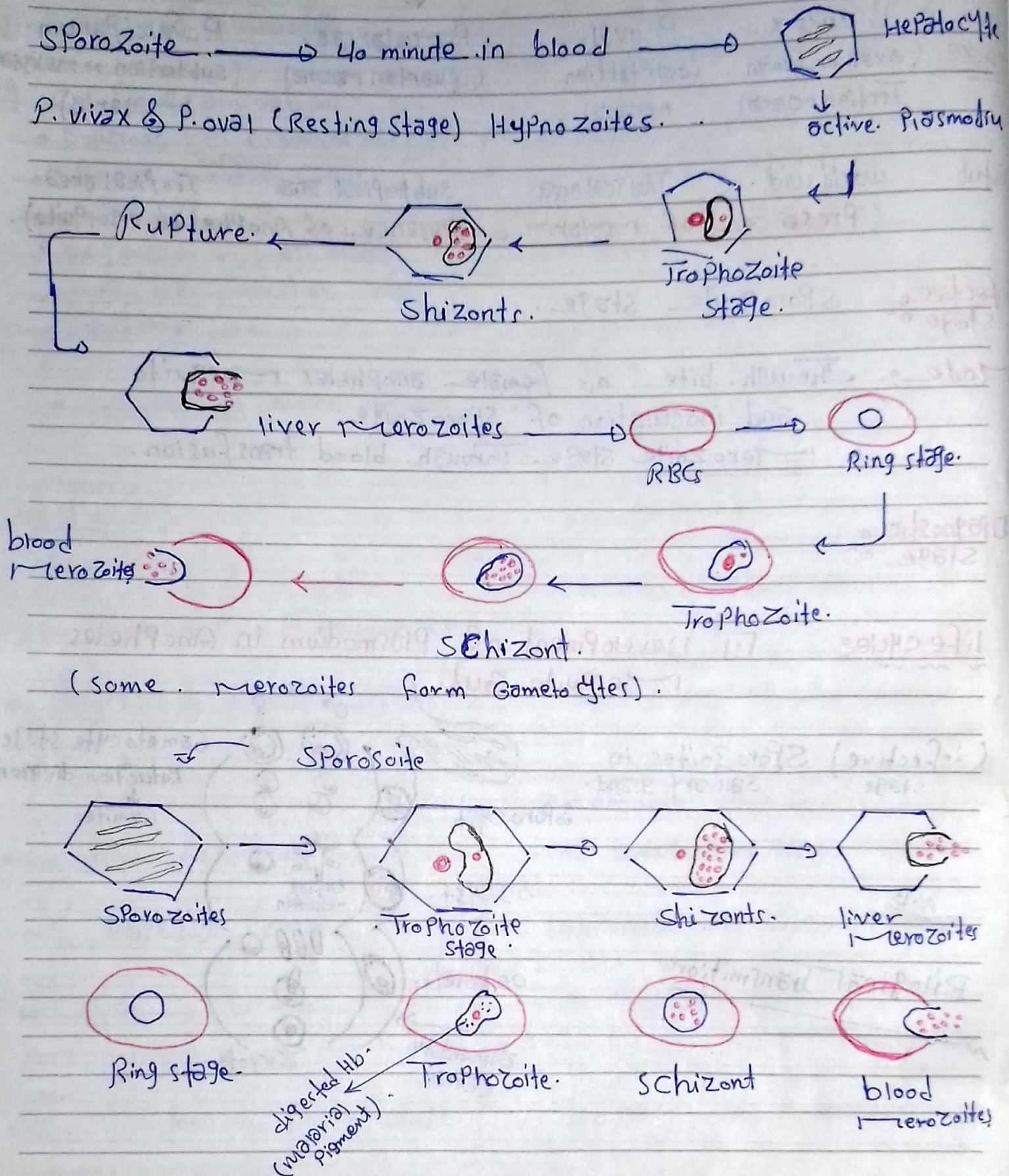
(infective) Sporozoites in Salivary gland.



N.B

Biological Transmission.

12] Development of Plasmodium in infective human



Pathogenesis and Clinical Picture

① Incubation Period followed by influenza like symptoms.

② Malaria Paroxysm (clinical attack).

* due to :-

- ✓ Rupture of RBCs.
- ✓ Release of Parasites & metabolites.
- ✓ Immuno logic Response.

* it include :- (3 stages)

① Cold stage :-

sudden chill, External cold & Temperature is Elevated.
lasts for 15 minute.

② Hot stage

Headache & High Fever & Hot & dry, flushed skin.
lasts 2-6 hours.

③ sweating stage :-

Profuse sweating.
Falls temperature.
Patient is weak & exhausted.

N.B Malaria Paroxysms is Repeated as follow.

- Every 3 days in P. Vivax & P. oval
- Every 4 days in P. Malariae.
- irregular in P. falciparum.

* it is Repeated for 2 weeks with ↓ intensity
Then Stop.

Date :

Short
 → Recurrence of clinical attack is called **Relapse** in P. vivax & P. oval Due to ?
 Reactivation of hypnozoites in liver.

→ Recurrence of clinical attack is called **Recrudescence** in P. malariae & P. falciparum due to ??
 Persistence of low grade Parasitaemia when the patient becomes debilitated.

[3] **Anemia due to destruction of RBCs.**

- P. vivax & P. oval invade ^(young RBCs) Reticulocyte of RBCs.
 - P. malariae invade old RBCs only.
 - P. falciparum invade any age of RBCs.
- Restricted Anemia. → severe anemia.

[5] **Enlarged liver & spleen.**

Due to enhanced phagocytosis of remnants of ruptured red cells and debris of schizont.

Complication :-

- ① P. vivax & P. oval & Malariae Malaria are Relatively benign.
- ② chronic P. Malariae infection. Result in nephrotic syndrome.
Result in immuno-complex deposition on glomerular wall.
- ③ P. falciparum is usually sever and fatal. Due to.
 - (a) infected RBCs develop knobs on its surface. so adhere to specific Receptor present in Endothelium cell of small Capillary → Partial occlusion. → anoxia, Necrosis

Clinical Picture differ according to site of occlusion.

- Cerebral Malaria :- sever headache, drowsiness, Coma Confusion.
 - Dysentric Malaria :- abdominal pain, Vomting, bleeding, diarrhea, dysentery.
 - Pulmonary edema :- ↓ blood flow in Pulmonary Circulation
 - hypoglycemia :- Result from impaired hepatic Gluconeogenesis
 - Renal failure :- Renal anoxia → acute Renal failure.
- ⑥ Hyper active Malaria Splenomegaly :-
Spleen markedly Enlarged with increases IgM Production.
due to Reduction of T-suppressor cell That Control B-cell activation.

⇐ (C) Black water Fever.

sheet

Black water Fever:-

Result from ① Repeated attack of *P. falciparum*
② incomplet quinine therapy.

characterized by

- ① massive intra vascular Haemolysis
- ② Anemia
- ③ jaundice.
- ④ Fever

causes:- auto immune with development anti body against infected RBCs.

Malaria pigment = Haemozoin.

Date :

Page :

Diagnosis :-

1] Clinical Manifestation.

2] Laboratory :-

a) Examination of Thin and Thick blood film. from the Patient. This show Ring, Trophozoite, schizont, gametocyte stage.

n.b in *P. falciparum* only Ring and gametocyte stage are seen??

As RBCs are Trapped in blood capillary of internal organ. due to surface knobs develop.

b) detection of circulating parasite Antigen using. Monoclonal antibody.

c) detection of Parasite DNA or RNA in Patient blood using. DNA and RNA Probes.

Schuffner dots

Ziemann's dots

Maurer's clefts

called stippling.

Degeneration Process occurs in.

Plasmodium infected RBCs.

Treatment of human malaria :-

- ① Drug destroy parasite stage in liver.
Pyrimethamine or Primaquine
- ② Drug destroy parasite stage in blood.
Chloroquine
- ③ Drug destroy gametocytes.
Primaquine, chloroquine.

⇒ Recommended for Treatment. (Recommended Regimen of anti malarial treatment)

- During clinical attack. chloroquine.
- Radical Treatment in case of P. vivax or P. oval.
Primaquine is given after treatment of clinical attack
to kill hypnozoite of P. vivax or P. oval.
- Drug Resistant Malaria is overcome by drug combination.
as Coartem
- Anti malarial chemoprophylaxis for healthy human.
entering an endemic area. as
 - Pyrimethamine or Primaquine.
 - Chloroquine.

Malaria

Primaquine,
chloroquine,
Pyrimethamine.

Toxoplasmosis

Spiramycin.

Trypanosoma

early: Pentamidine.
late: Trypanamide.
both:-



Human naturally resistant to malaria

1] Absence of Duffy Antigen

2] Haemoglobin S (in sickle-cell anemia).
shape of RBCs and type of Hb are not suitable for parasite growth.

3] Deficiency of G6PD.
as parasite need enzyme for growth

Control :-

- treatment of cases.
- Mosquito control.
- Chemo Prophylaxis.
- Vaccine against malaria.



Babesia Species

infective stage: Sporozoites.
 Vector: Hard Ticks.
 Diagnostic stage: Merozoite.
 Mode: Through bite of Hard tick.

Pathogenesis and clinical picture.

- 1] Asymptomatic.
- 2] Malaria like picture with hemolytic anemia but no periodicity.
- 3] Fulminant disease that may end fatally in.
 - splenectomized patient.
 - immunodeficient patient.

Diagnosis:

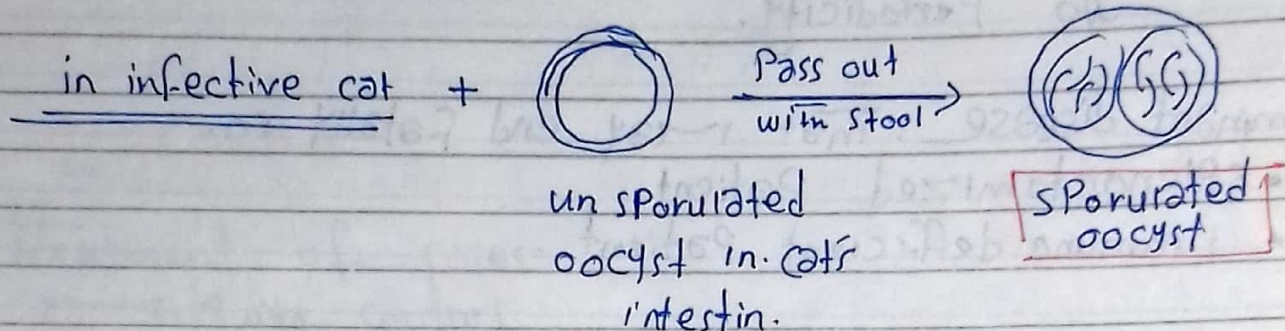
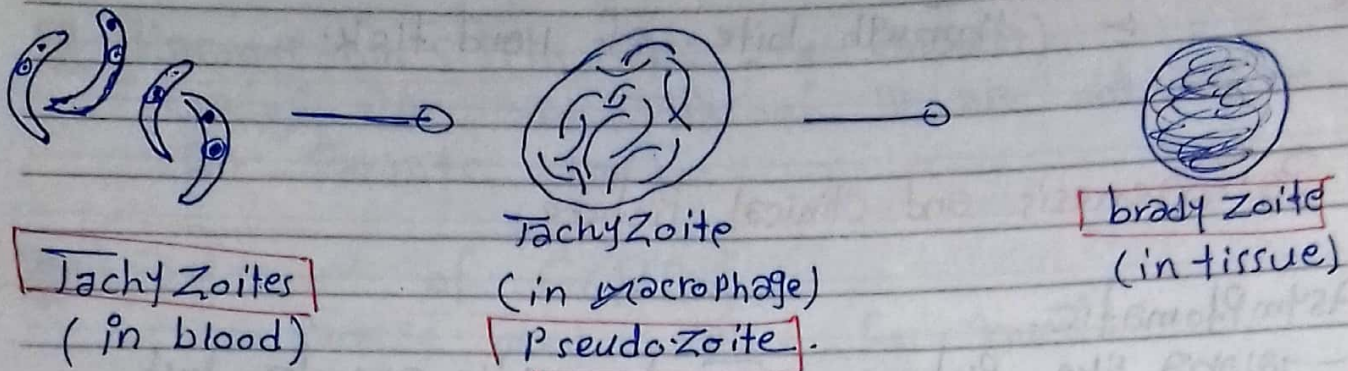
1. Blood film examination. $\Rightarrow$ Multiple small ring in RBCs. differentiated from *P. falciparum* by absence of malarial pigment.
2. Serology.
3. Detection of DNA of parasite by P.C.R.

Treatment.

- Combination of Clindamycin, Quinin.
- Blood Transfusion in severe case.

Toxoplasma gondiiToxoplasmosis

Distribution is world wide.



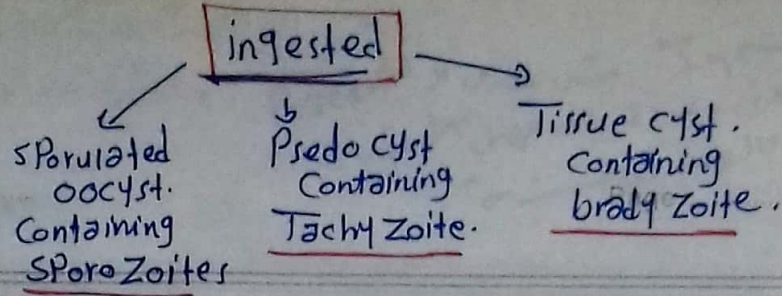
- Infective stage :-
- Tachyzoite
 - Pseudozoite
 - Tissue cyst
 - sporulated oocyst.

Definitive host :- cat.

Mode of infection :-

- ① ingestion of sporulated oocyst in contaminated food or drink.
- ② ingestion of infected under cooked meat containing Pseudocyst or tissue cyst
- ③ organ Transplantation containing Pseudo or tissue cyst.
- ④ Blood Transfusion containing tachy zoite.
- ⑤ Trans Placental ~ ~ ~
- ⑥ Contamination of m.m or skin abrasion.

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Pathogenesis and Clinical Picture

1] **Congenital infection** :- (congenital toxoplasmosis).
Depend on

- Protective immunity of mother.
- Age of foetus at Time of infection.

(a) **loss of Fetus** due to abortion or still birth.

(b) **Early neonatal manifestation** :-

→ **C.N.S manifestation** :-

- microcephaly
- hydrocephalus
- spasticity
- convulsion

→ **Eye manifestation** :-

Retino choroditis

→ **systemic manifestation** :-

- Fever
- Pneumonitis
- Hepatomegaly
- Jundice
- Lymphadenitis

(c) **late manifestation** :-

- ✓ C.N.S involvement and mental Retardation.
- ✓ Eye affection.

2] **Acquired toxoplasmosis**

Depend on.

- ✓ immune state of infected person.
- ✓ Age of infected person.
- ✓ Virulence of some strain of toxoplasma

- * Asymptomatic.
- * Lymphadenitis, fever, headch, skin Rash, Splenomegally.
- * Retino choroditis it can Result in blind ness.

③ Toxoplasmosis in immune Compromized Patient.

- ✓ Encephalitis it is the most common manifestation.
Due to Reactivation of cerebral cyst
- ✓ organ Trans plant* can develop acute Toxoplasmosis.

Diagnosis

1- Clinical :- according to clinical manifestation.

2. Radiology :-

* X-Ray. → show calcification.

ultra sound → detect Enlargment of ventricle.

3. laboratory.

* Serology :-

✓ Detection of IgM in Patient's blood indicate to Recent infection.

✓ Detection of IgG (Rising titter) indicate to Active infection.

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✓ Sabin Feldman dye test.

- serum of Patient added to toxoplasma organism.
- Added Methylene blue.
- if serum contain Antibodies The organism not take the dye
- if serum not contain Antibodies The organism Take the dye → a negative Reaction.

✓ **Frenkle test.** (Toxoplasmin intradermal test.).

✓ **Molecular Techniques:**

Detection of Parasite DNA by P.C.R.

Diagnosis of Congenital Toxoplasmosis.

1. Detection of IgM in blood of baby indicates foetal infection.
(Maternal IgM doesn't cross Placenta).
2. Detection of Parasite DNA in Amniotic fluid of infants

Treatment:

- Combination of Pyrimethamine + Tri sulpho Pyrimidine
- **Spiramycin** (to infected pregnant women)

